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Thrombus aspiration is associated with improved platelet inhibition rate following dual antiplatelet therapy in acute myocardial infarction patients

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Abstract

Background It is well-established that thrombus aspiration during primary percutaneous coronary intervention (PCI) in patients with acute myocardial infarction (AMI) indicates a higher thrombus burden and necessitates more intensive antithrombotic therapy. The bidirectional association between adverse events in AMI patients and platelet reactivity is typically observed during dual antiplatelet therapy (DAPT).

Objective To investigate platelet reactivity after DAPT in AMI patients with thrombus aspiration performed during PCI.

Methods In this retrospective study, we examined 269 consecutive AMI patients who underwent PCI and recorded their demographic, clinical and laboratory data. The platelet reactivity was measured with thromboelastogram (TEM).

Results Ultimately, 208 patients were included in this study and divided into a Thrombus Aspiration group (N = 97) and a PCI Alone group (N = 111) based on whether thrombus aspiration was performed or not. The adenosine diphosphate (ADP)-induced platelet inhibition rate in the Thrombus Aspiration group was higher than that in the PCI Alone group ($P < 0.001$). Furthermore, multivariate linear regression analysis revealed that the ADP-induced platelet inhibition rate was independently associated with leukocyte count, thrombus aspiration and the combination of aspirin and ticagrelor as DAPT after adjusting for potential covariates in all AMI patients.

Conclusion In conclusion, clinicians should exercise heightened attention towards the bleeding risk among patients undergoing PCI concomitant with Thrombus Aspiration postoperatively.

Keywords Myocardial infarction, Platelet antagonist, Percutaneous coronary intervention, Platelet function tests, Thrombosis

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Introduction

Based on existing evidence, it is indicated that the underlying pathophysiology of most cases of acute myocardial infarction (AMI) is associated with thrombosis related to atheromatous plaques [1]. The standard treatment for patients with acute myocardial infarction (AMI), especially those who undergo percutaneous coronary intervention (PCI), involves dual antiplatelet therapy (DAPT) [2]. This therapy combines the use of aspirin and a P2Y₁₂ receptor antagonist to prevent the reoccurrence of thromboembolic events.

For AMI patients with a higher thrombus burden, interventional cardiologists typically opt for thrombus aspiration as a standalone procedure or combine it with balloon angioplasty and/or stent implantation during PCI [3, 4]. Clinically, compared to those without thrombus aspiration, AMI patients undergoing thrombus aspiration typically receive more intensive antithrombotic treatment after PCI, in addition to DAPT. This may involve a prolonged duration of low-molecular-weight heparin injection and/or the addition of glycoprotein IIb/IIIa antagonists [5]. However, excessive use of antithrombotic agents poses an increased risk of bleeding events, which may be life-threatening in some cases [6]. Therefore, it is crucial for judiciously administer antiplatelet agents, maintaining platelet reactivity within a safe and effective range for AMI patients.

In recent years, various methods have been developed to measure platelet function and assess the efficacy of antithrombotic agents [7]. Among these, thromboelastography (TEM) has emerged as a new platelet function test that is more accurate and reliable [8]. It can measure the actual platelet inhibitory effect of aspirin and ADP receptor antagonists, even when these agents taken in combination.

Nonetheless, whether AMI patients undergoing thrombus aspiration exhibit a higher platelet reactivity after standard DAPT, potentially increasing the risk of future thrombotic events, has not been comprehensively investigated. Herein, we sought out to examine platelet reactivity in AMI patients with and without thrombus aspiration after DAPT in our center.

Methods

Study population

This retrospective cohort study examines a cohort of 269 AMI patients who underwent percutaneous coronary intervention (PCI) and were admitted to the Cardiac Care Unit (CCU) at Xinhua Hospital, affiliated with Shanghai Jiao Tong University School of Medicine, from January 2022 to December 2023. The study's protocol adhered to the principles of the Declaration of Helsinki. Approval for this study was obtained from the Ethics Committee of

Xinhua Hospital affiliated to Shanghai Jiao Tong University School of Medicine (No. XHEC-D-2024-005).

Medication and procedure protocol

A loading dose of 300 mg aspirin and either 300 mg or 600 mg clopidogrel or 180 mg ticagrelor, was administered before PCI. Maintenance doses of DAPT consisted of 100 mg aspirin per day and either 75 mg clopidogrel per day or 90 mg ticagrelor twice a day. The decision to perform thrombus aspiration during PCI was made based on the experience of interventional cardiologists and in accordance with relevant relative guidelines.

Clinical data collection

Complete blood count, liver function, renal function, blood lipid levels, fasting glucose, glycosylated hemoglobin, and hypersensitivity C-reactive protein were assessed with fasting blood samples on the morning of following admission. An intracoronary thrombus was detected through angiography and assessed using a five-point grading system, in accordance with previous research. Following the reclassification of the thrombus and guided by clinical data, 3–4 grades in the classification are defined as high thrombus [9].

Platelet function measurement

TEM was conducted at least 5 days, and at most 7 days, after the initiation of DAPT. Whole blood samples were collected in vacutainer tubes containing 3.2% trisodium citrate, and the initial 2–4 ml of blood was discarded to avoid platelet activation. The TEG assay was performed according to the manufacturer's instructions, measuring the antiplatelet effect through the adenosine diphosphate (ADP) or arachidonic acid (AA) pathway. The percentage of platelet inhibition was calculated using computer software based on the formula: $\text{ADP or AA-induced platelet inhibition (\%)} = 100 - 100 \times [(MA_{AA \text{ or ADP}} - MA_{\text{Fibrin}}) / (MA_{\text{Thrombin}} - MA_{\text{Fibrin}})] \times 100\%$. Where, MA_{ADP} represents adenosine diphosphate-induced clot strength, MA_{FIBRIN} is FIBRIN-induced clot strength, and MA_{THROMBIN} is THROMBIN-induced clot strength.

Statistical analysis

Continuous variables were presented as mean \pm standard deviation for variables following a normal distribution and median (interquartile range) for data with skewed distributions. Continuous variables were compared using the Student's *t*-test or Mann–Whitney *U* test. Categorical variables were expressed as numbers (percentages) and were compared using the chi-square (χ^2) test or Fisher's exact test. Normality was assessed using the Kolmogorov–Smirnov test. ADP-induced platelet inhibition (ADP%) was subjected to a normal transformation

to ADP%' for multivariate linear regression analysis: $ADP\% = \sqrt{K - ADP\%}$, where $K = (ADP\%)_{max} + 1$. Univariate analysis identified variables with $P < 0.1$, considered significant, which were then included in multivariate linear regression analysis. Adjustments were made for seven variables, including age, male, baseline hemoglobin level, total cholesterol, leukocyte count, thrombus aspiration, and the combination of aspirin and ticagrelor. During multivariate linear regression analysis, the stepwise elimination method was used to identify independent factors associated with ADP-induced platelet inhibition. A two-sided P -value < 0.05 was statistically significant, and all data analyses were performed using SPSS software version 26.0.

Results

Baseline characteristics

Cases that did not receive DAPT (n=15), those lacking TEG results (n=26), and those with incomplete data (n=4) were excluded. Another 16 patients were excluded based on additional exclusion criteria, including severe anemia, malignant disease, serious renal or hepatic insufficiency, and a total platelet count $< 100 \times 10^9 /L$. Finally, 208 patients were enrolled in this study (Fig. 1).

A total of 208 patients were included in this retrospective study, exhibiting male predominance (n=168, 80.8%) with a mean age of 65.5 ± 11.5 years. The Thrombus Aspiration group comprised 97 (46.6%) patients, while the PCI Alone group included 111 (53.3%) patients.

Compared to the PCI Alone group, patients in the Thrombus Aspiration group were significantly younger ($P < 0.001$), with a higher proportion of males ($P = 0.002$) and higher levels of hemoglobin ($P < 0.001$), mean platelet volume ($P = 0.014$), and alanine transaminase ($P < 0.001$). Additionally, the Thrombus Aspiration group comprised significantly more patients treated with aspirin and

ticagrelor ($P < 0.001$) and a higher prevalence of patients with ST-elevation myocardial infarction (STEMI) type ($P = 0.005$). No significant differences were observed in other indicators between the two groups (Table 1, Fig. 2).

TEG analysis

In comparison to the PCI Alone group, the thrombus aspiration group demonstrated a significantly higher ADP-induced platelet inhibition rate ($P < 0.001$). However, there was no significant difference in the AA-induced platelet inhibition rate between these two groups ($P > 0.05$) (Table 2, Fig. 2).

Multivariate analysis for ADP-induced platelet inhibition rate in PCI patients

During multivariate analysis with ADP-induced platelet inhibition rate as the dependent variable, significant factors identified during the univariate analysis (age, male, baseline hemoglobin level, leukocyte count, thrombus aspiration, high thrombus and the combination of aspirin and ticagrelor with $P < 0.1$) were utilized as independent variables. The results of the multivariate regression analysis indicated that a higher ADP-induced platelet inhibition rate independently correlated with three factors: lower leukocyte count ($P = 0.01$), thrombus aspiration ($P < 0.001$), and the combination of aspirin and ticagrelor ($P < 0.001$) (Table 3).

Discussion

In this study, we used TEG to evaluate platelet reactivity and examine the efficacy of DAPT in AMI patients undergoing PCI and thrombus aspiration. In addition, the determinants of platelet reactivity in AMI patients were investigated. Contrary to our expectations, AMI patients subjected to thrombus aspiration exhibited a higher ADP-induced platelet inhibition rate after DAPT.

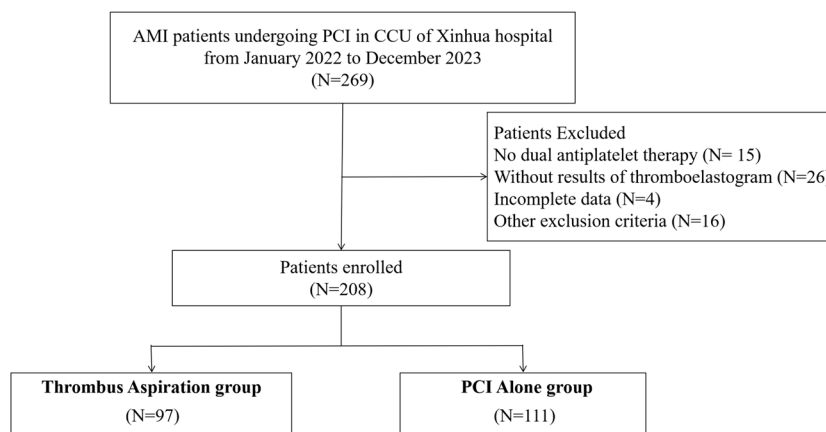


Fig. 1 Flowchart of the study. AMI acute myocardial infarction, CCU cardiac care unit, PCI percutaneous coronary intervention

Table 1 Baseline clinical and laboratory characteristics of AMI patients

Variables	Thrombus aspiration (N = 97)	PCI alone (N = 111)	P-value
Age, year	61 (57,69)	70 (62,76)	<0.001
Male, n (%)	89 (91.8)	79 (71.2)	<0.001
Smoking, n (%)	57 (58.8)	53 (47.7)	0.112
LVEF, %	53 (45.7,58.9)	54.3 (49.5,59.1)	0.257
Hypertension, n (%)	67 (69.1)	75 (67.6)	0.816
Diabetes mellitus, n (%)	35 (36.1)	48 (43.2)	0.293
Leukocyte count, 10 ⁹ /L	9.21 (8.0,11.0)	9.4 (7.7,11.2)	0.846
Neutrophil count, 10 ⁹ /L	7.8 (6.1,9.5)	7 (5.6,8.9)	0.076
Hemoglobin, g/L	140.5 ± 12.9	128.3 ± 17.6	<0.001
Platelet, 10 ⁹ /L	215 (187,245)	210 (183,250)	0.933
MPV, fL	10.1 (9.35,10.8)	9.7 (8.7,10.6)	0.014
BUN (mmol/L)	6 (5,7.4)	6.3 (5,8.1)	0.155
Serum creatinine (μmol/L)	79 (67.1,93.5)	86 (68,110.4)	0.066
Uric acid (μmol/L)	378.6 ± 109	383.8 ± 127.1	0.753
ALT, U/L	44 (31,68.5)	29 (17,46)	<0.001
TC, mmol/L	4.5 (3.8,5.4)	4.5 (3.8,5.3)	0.806
Triglyceride, mmol/L	1.6 (1.1,2.2)	1.4 (1.1,1.8)	0.077
HDL-C, mmol/L	1 ± 0.2	1.0 ± 0.2	0.262
LDL-C, mmol/L	2.91 (2.4,3.5)	2.8 (2.3,3.5)	0.35
Apolipoprotein A, g/l	1.1(0.95,1.2)	1.1 (0.94,1.26)	0.47
Glucose, mmol/L	6.8 (5.4, 8.4)	6.6 (5.4,8.1)	0.969
Hs-CRP, mg/L	10.86 (9.14, 26.1)	17.8 (9.3,35.2)	0.052
HbA1c	6.6 (5.8, 7.5)	6.7 (5.9,7.8)	0.532
Aspirin + Ticagrelor, n (%)	87 (89.7)	69 (62.2)	<0.001
STEMI, n (%)	73 (75.3)	63 (56.8)	0.005
High Thrombus, n (%)	94 (96.9)	104 (93.7)	0.28
TIMI flow grade (final) < 3, n (%)	6 (6.1)	8 (7.2)	0.769

The data are presented as mean ± standard deviation for continuous variables, median (interquartile range) for variables with a skewed distribution, or n (%) for categorical variables. ALT alanine transaminase, BUN blood urea nitrogen, HbA1c glycosylated haemoglobin, HDL-C high-density lipoprotein cholesterol, Hs-CRP high sensitivity-C reactive protein, LDL-C low-density lipoprotein cholesterol, LVEF left ventricular ejection fraction, MPV mean platelet volume, STEMI ST-elevation myocardial infarction, TC total cholesterol.

Besides, AMI patients with an elevated ADP-induced platelet inhibition rate demonstrated a higher baseline leukocyte count, more frequent thrombus aspiration, and a higher prevalence of treatment with the combination of aspirin and ticagrelor as DAPT.

AMI stands as one of the most life-threatening cardiovascular diseases globally, with thrombosis affecting over 90% of AMI patients [1]. PCI is the preferred method for achieving rapid coronary reperfusion in AMI patients, though it introduces the risk of distal embolization from proximal thrombotic particles and/or atherosclerotic plaque debris during guidewire passage through the occlusive lesion. Thrombus burden as an independent predictor of adverse cardiovascular events in STEMI patients underscores the importance of thrombus aspiration as a viable intervention characterized by high thrombus burden [10]. Although not recommended as a routine procedure, the European Society of Cardiology

(ESC) 2017 guidelines suggest thrombus aspiration in the management of AMI patients with elevated thrombus burden to mitigate the risk of distal embolization and preserve coronary microcirculation [4, 11]. Recent findings by Elfekky et al. consistently highlighted the significant value of thrombus aspiration in reducing in-hospital mortality and improving various cardiovascular parameters in STEMI patients with high thrombus burden, compared to those undergoing PCI alone, as observed over a 1-month follow-up period [12].

Given the pivotal role of platelets in thrombosis, the evaluation of platelet activity has attracted significant interest [13]. Over the years, several platelet function tests, such as light transmission platelet aggregation (LTA), Vasodilator-Stimulated Phosphoprotein (VASP) index, VerifyNow P2Y12 assay, and TEG, have been employed to assess individual responses to antiplatelet therapy [8, 14, 15]. LTA is commonly seen as the gold

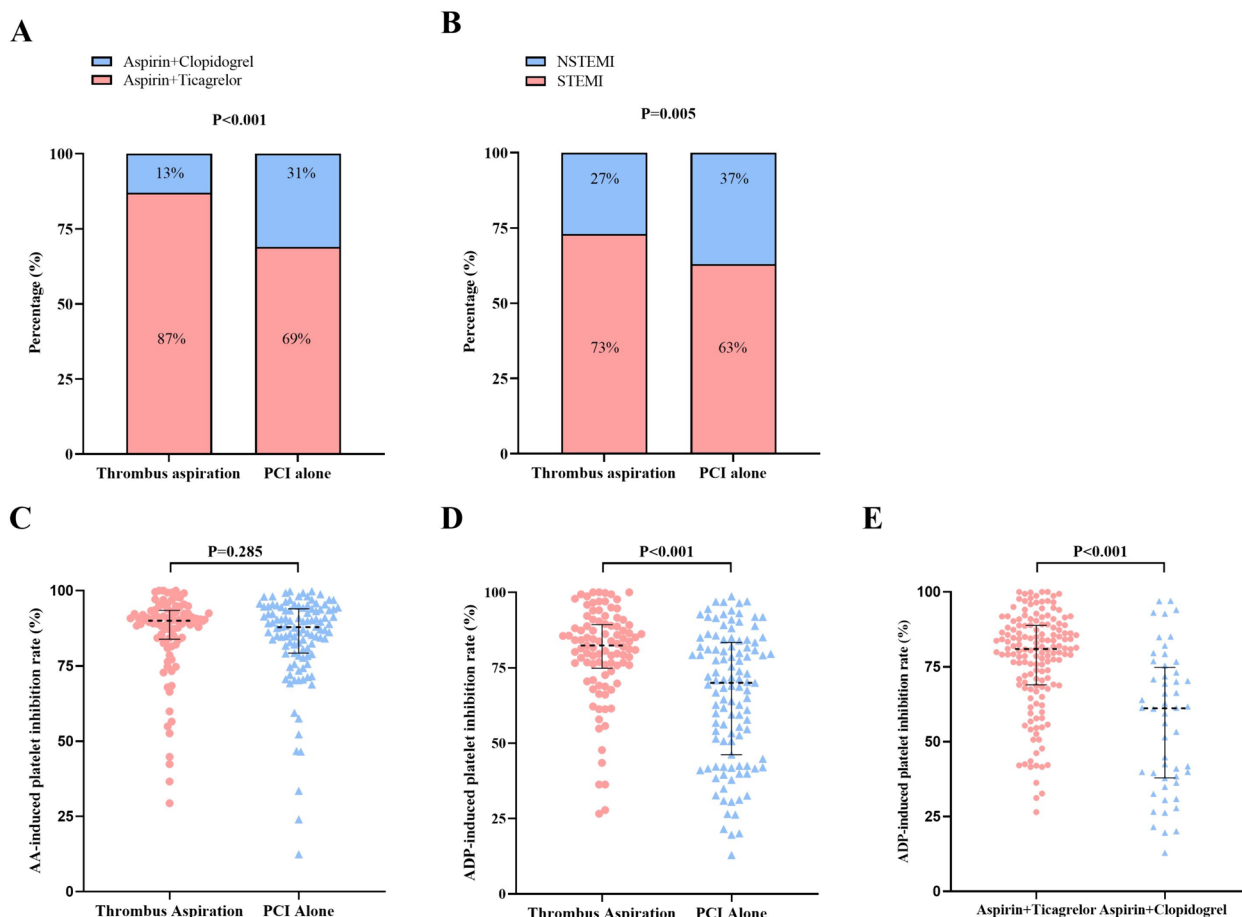


Fig. 2 Comparison of medication (A), MI Type (B), AA-induced platelet inhibition rate (C), and ADP-induced platelet inhibition rate (D) between the thrombus aspiration and PCI alone groups. E Comparison of ADP-induced platelet inhibition rate between AMI patients using aspirin + clopidogrel and aspirin + ticagrelor. P represents the comparison of the proportion of STEMI type or the combination of aspirin and ticagrelor between the two groups in (A) and (B). The dotted line in the middle represents the median, while the upper and lower solid lines denote quartiles in (C), (D), and (E). AA arachidonic acid, ADP adenosine diphosphate, NSTEMI non-ST-segment elevation myocardial infarction, STEMI ST-segment elevation myocardial infarction

Table 2 Antiplatelet effect evaluated by thromboelastography

Variables	Thrombus aspiration (N = 97)	PCI alone (N = 111)	P-value
AA-induced platelet inhibition rate (%)	90 (83.9,93.5)	87.9 (79.3,94)	0.285
ADP-induced platelet inhibition rate (%)	82.4 (74.9,89.4)	70 (46.2,83.4)	< 0.001

The data are expressed as median (interquartile range). AA arachidonic acid, ADP adenosine diphosphate, PCI percutaneous coronary intervention

standard for platelet function testing, but its clinical use is limited due to drawbacks like time-consuming detection, complex sample preparation, and low specificity for

P2Y12 antagonists [14]. It is now understood that ticagrelor, in addition to inhibiting the P2Y12-ADP pathway, can reduce platelet reactivity by enhancing adenosine activation through increased adenosine levels and subsequent engagement of adenosine A2A receptors [16]. Hence, traditional assays like the VASP index and VerifyNow P2Y12, which focus solely on the P2Y12-ADP pathway, may not accurately capture the overall antiplatelet effect of ticagrelor. TEG has emerged as a novel platelet function test, providing rapid and precise platelet aggregation detection without the constraints of other methods [17]. However, there is no clear international consensus on the "safety range" of ADP% in TEG. It has been shown that an ADP% greater than 30 indicates effective drug action, while an ADP% greater than 92.5 is an independent predictor of bleeding, though these data are specific to clopidogrel [18–20]. Whether different types

Table 3 Clinical parameters associated with normally converted ADP-induced platelet inhibition rate

Variables	Univariate			Multivariate (stepwise elimination)		
	Estimate, 95% CI	SEM	P value	Estimate, 95% CI	SEM	P value
Age	0.042 (0.019,0.064)	0.011	<0.001			
Male	-0.801 (-1.461, -1.141)	0.335	0.018			
Smoking	-0.282 (-0.808,0.245)	0.267	0.293			
LVEF, %	-0.009 (-0.041,0.022)	0.016	0.555			
Hypertension	0.133 (-0.433,0.699)	0.287	0.645			
Diabetes mellitus	0.417 (-0.118,0.952)	0.271	0.126			
Leukocyte count	-0.056 (-0.111,0.001)	0.028	0.052	-0.059 (-0.104, -0.014)	0.023	0.01
Neutrophil	-0.069 (-0.158,0.019)	0.045	0.125			
Hemoglobin	-0.024 (-0.039, -0.008)	0.008	0.003			
Platelet	-0.003 (-0.008,0.002)	0.003	0.31			
MPV	-0.066 (-0.248,0.115)	0.092	0.472			
BUN	-0.001 (-0.082,0.081)	0.041	0.998			
Serum creatinine	0.003 (-0.003,0.009)	0.003	0.306			
Uric acid	0.001 (-0.003,0.002)	0.001	0.745			
ALT	-0.006 (-0.014,0.001)	0.004	0.105			
TC	-0.160 (-0.362, 0.041)	0.102	0.118			
Triglyceride	-0.288 (-0.633,0.057)	0.175	0.101			
HDL-C	-0.228 (-1.449,1.994)	0.619	0.714			
LDL-C	-0.222 (-0.494,0.051)	0.138	0.11			
Apolipoprotein A	-0.203 (-1.373,0.968)	0.594	0.733			
Glucose	0.008 (-0.096,0.113)	0.053	0.875			
Hs-CRP	-0.002 (-0.009,0.005)	0.004	0.564			
HbA1c	0.123 (-0.055,0.301)	0.090	0.175			
STEMI	-0.316 (-0.868,0.236)	0.280	0.261			
Thrombus aspiration	-1.325 (-1.821, -0.829)	0.252	<0.001	-0.94 (-1.428, -0.453)	0.247	<0.001
Aspirin + ticagrelor	-1.814 (-2.369, -1.258)	0.282	<0.001	-1.497 (-2.058, -0.936)	0.285	<0.001
High thrombus	-1.069 (-2.293, 0.154)	0.62	0.086			

Multivariable regression analyses were conducted with adjustments for variables that demonstrated significance ($P < 0.1$) in univariable analysis, including age, gender, baseline hemoglobin level, leukocyte count, thrombus aspiration, high thrombus and the combination of aspirin and ticagrelor

ADP adenosine diphosphate, ALT alanine transaminase, BUN blood urea nitrogen, HbA1c glycosylated haemoglobin, HDL-C high-density lipoprotein cholesterol, Hs-CRP high sensitivity-C reactive protein, LDL-C low-density lipoprotein cholesterol, LVEF left ventricular ejection fraction, MPV mean platelet volume, STEMI ST-elevation myocardial infarction, TC total cholesterol

of P2Y12 receptor inhibitors share the same "safety margins" remains to be further explored. Recently, a study on DAPT treatment in stroke patients with ticagrelor suggested a recommended ADP% safety range of 30–82.3% [21]. In our study, the ADP% in the Thrombus Aspiration group is approximately 80.3%. Therefore, clinicians should be particularly vigilant regarding the bleeding risk in PCI patients with Thrombus Aspiration undergoing DAPT.

The mean platelet volume (MPV) is recognized as an indicator of platelet size and, consequently, platelet reactivity [22]. Lai et al. demonstrated that elevated mean platelet volume at admission was an independent predictor of a large thrombus burden in STEMI patients, consistent with the findings of our study [23]. Notably,

in this study, the Thrombus Aspiration group exhibited a higher mean platelet volume and comprised a significantly higher percentage of patients with STEMI. STEMI typically indicates complete coronary artery occlusion with thrombosis and aligns with the pathophysiology of type 1 myocardial infarction (MI) according to the fourth universal definition of myocardial infarction. Conversely, non-ST-elevation myocardial infarction (NSTEMI) often indicates incomplete coronary artery occlusion, with the pathophysiology of NSTEMI tending to be more diverse. The observed correlation between higher mean platelet volume, ST-segment elevation, and a higher percentage of STEMI patients in the Thrombus Aspiration group suggests an association between elevated platelet volume and increased thrombus burden in AMI patients.

Contrary to expectations, we found that AMI patients with a higher thrombus burden, indicated by thrombus aspiration during the PCI procedure, experienced more significant ADP-induced platelet inhibition compared to those with a lower thrombus burden. Meanwhile, the Thrombus Aspiration group in our study showed a significantly higher percentage of ticagrelor usage compared to the PCI Alone group. Clopidogrel and ticagrelor, both P2Y₁₂ receptor antagonists, differ in their mechanisms of action. Clopidogrel, a prodrug, undergoes liver metabolism via CYP2C19, which, when genetically variant (25–35% of population), may cause variable drug response and resistance [24]. In contrast, ticagrelor, not requiring hepatic metabolism, binds reversibly to the P2Y₁₂ receptor, resulting in a consistently faster, more potent, and less variable antiplatelet effect compared to clopidogrel in clinical studies [9]. Our study's findings, exemplified by the higher antiplatelet effect of ticagrelor in AMI patients compared to clopidogrel (Fig. 2E), align with previous research. However, numerous studies have demonstrated that, compared to clopidogrel, the use of ticagrelor is significantly associated with a higher risk of bleeding in Chinese patients [25]. The increased proportion of ticagrelor usage also reflected clinicians' consideration that these AMI patients might benefit from more intensive antithrombotic therapy.

To investigate factors impacting platelet reactivity, particularly to ascertain whether the elevated ADP-induced platelet inhibition observed in the Thrombus Aspiration group resulted from increased ticagrelor usage, we conducted a multiple linear regression analysis of ADP-induced platelet inhibition rates. The outcomes revealed that ADP-induced platelet inhibition was independently associated with thrombus aspiration. Furthermore, heightened leukocyte counts and the administration of ticagrelor independently correlated with increased ADP-induced platelet inhibition. It is widely acknowledged that in the intricate pathogenesis of coronary atherosclerotic disease (CAD), inflammation assumes a pivotal role [26]. The activation of inflammatory cells within atherosclerotic plaques renders them unstable, fostering thrombotic events. A heightened leukocyte count, as an inflammatory marker, not only signals a graver prognosis and elevated mortality in CAD patients, but is also a constituent of the "PRECISE-DAPT" score [27]. Widely employed to assess bleeding risk in AMI patients and guide the duration of DAPT post-stent implantation, the "PRECISE-DAPT" score incorporates leukocyte count as a contributing factor [28]. This study revealed that AMI patients with an elevated leukocyte count exhibited a heightened ADP-platelet inhibition rate after DAPT. This is in accordance with the predictive function of bleeding risk in the "PRECISE-DAPT" score and also potentially

elucidates the mechanism behind bleeding risks during DAPT. Controversies persist regarding the relationship between leukocyte count and the antiplatelet effect in CAD patients. A recent study by Li et al. uncovered that the leukocyte count was an independent predictor of low platelet reactivity in PCI patients undergoing DAPT [29]. These discrepancies may primarily stem from variations in antiplatelet medications and participant demographics. Li et al.'s study, limited to clopidogrel treatment and encompassing participants beyond AMI patients, contrasts with our study's focus.

Despite our comprehensive investigation, the correlation between thrombus aspiration and post-DAPT platelet reactivity in AMI patients remains elusive. It is highly conceivable that thrombus aspiration is not the causative factor for the heightened ADP-induced platelet inhibitory effects observed in AMI patients undergoing DAPT; rather, it is a distinctive characteristic. AMI patients with a higher thrombus burden and elevated leukocyte count are predisposed to an increased bleeding risk after DAPT, particularly with ticagrelor treatment. This subset of patients exhibits a propensity for a more pronounced platelet inhibitory effect. TEM has emerged as a valuable tool for assessing bleeding risk in these individuals. Indeed, its application can guide clinicians in evaluating bleeding risk, prompting adjustments in medical dosage, shortening DAPT duration, and implementing specific precautions.

Despite these promising findings, certain limitations of this study should be acknowledged. Firstly, being a retrospective study conducted at a single center, there may be constraints on the applicability of the results to broader populations. Secondly, the relatively small sample size introduces the possibility of deviations in statistical outcomes, impacting the study's representativeness and reliability. Consequently, multi-centered larger-scale randomized clinical trials are imperative to conclusively confirm the relationship between thrombus aspiration and platelet inhibition rate. Thirdly, the mechanisms underlying thrombus burden, leukocyte count, and the efficacy of antiplatelet therapy remain unclear, necessitating further in-depth mechanistic experiments.

Conclusion

In the present study, thrombus aspiration is independently associated with a higher ADP-induced platelet inhibition rate after DAPT in AMI patients. Attention should be directed towards AMI patients undergoing thrombus aspiration, exhibiting elevated leukocyte counts, and receiving ticagrelor, as they are prone to a bleed risk following antithrombotic therapy.

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Author contributions

Qianyi Li, Chunxuan Wu wrote the manuscript. Chunxuan Wu, Yanyan Li, Juan Ma, Shiqun Sun and Xiaoxing Xu collected the data. Lingchao Yang and Ping Li analyzed the data. Ying Yu and Wei Li modified the manuscript. All authors read and approved the final version of the manuscript.

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Availability of data and materials

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Approval for this study was obtained from the Ethics Committee of Xinhua Hospital affiliated to Shanghai Jiao Tong University School of Medicine (No. XHEC-D-2024-005).

Competing interests

The authors declare no competing interests.

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